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Alcohol use and risk of non-Hodgkin's lymphoma among Connecticut women (United States)

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Abstract

Objective: Incidence rates of non-Hodgkin's lymphoma (NHL) have risen dramatically over the past several decades; however, the etiology of NHL remains largely unknown. Previous studies of the relationship between alcohol consumption and NHL have yielded conflicting results. Data from a population-based case–control study among women in Connecticut were analyzed to determine the potential impact of alcohol consumption on risk of NHL.
Methods: The study included 601 histologically confirmed, incident cases of NHL and 718 population-based controls. In-person interviews were administered using standardized, structured questionnaires to collect data on history of consumption for beer, wine, and liquor.
Results: When compared to non-drinkers, women who reported consumption of at least 12 drinks per year of any type of alcohol experienced slightly reduced risk of NHL (OR: 0.82; 95% CI: 0.65–1.04). Further stratification by alcohol type revealed that the inverse association was mainly limited to wine consumption (OR: 0.75; 95% CI: 0.59–0.96), with no clear association for beer or liquor consumption. Risk of NHL was further reduced with increasing duration of wine consumption (p for linear trend = 0.02). Consumption of wine for greater than 40 years was associated with approximately 40% reduction in risk (OR: 0.63; 95% CI: 0.44–0.91).
Conclusion: Our results are consistent with several recent epidemiologic studies that have also suggested an inverse association between wine consumption and risk of NHL. The reduction in risk of NHL associated with increased duration of wine consumption warrants further investigation in other populations.

Introduction

Non-Hodgkin's lymphomas (NHL) represent a heterogeneous group of lymphomas arising from the malignant transformation of lymphoid cells throughout the body [1]. Over the past several decades, age-adjusted incidence rates of NHL have risen steadily in the United States and throughout the world [2]. In Connecticut, NHL has become the fifth most common cancer among both men and women [3]. Despite these dramatic increases in NHL incidence, the etiology of NHL remains largely unknown.

A number of epidemiologic studies have investigated the potential impact of alcohol consumption on the risk of NHL [4–15]. The results of these studies, however, have been inconsistent. Two studies suggested a positive association among men, particularly those drinking beer and smoking cigarettes in a small cohort study [8], and those with a family history of NHL in a population-based case–control study [15]. In addition, there have been six reports of no association between alcohol consumption and risk of NHL in hospital-based case–control

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studies [4–6, 10, 11, 13]. However, inverse associations between alcohol consumption and risk of NHL have been reported in two population-based case–control studies for men [7, 14], and in one population-based case–control study [9] and one cohort study [12] for women.

In four studies, wine consumption was hypothesized to be inversely associated with risk of NHL [9, 11, 12, 14]. Of the epidemiologic studies that have considered the association between alcohol consumption and NHL, over half have evaluated the subtype of alcohol consumed [4, 7–9, 11–14], although only two evaluated the impact of each alcohol subtype while controlling for other alcohol subtypes [11, 14].

Based on the inconsistencies of previous research, we used the data from a population-based case–control study conducted among women in Connecticut to evaluate the impact of consumption of beer, wine, and liquor on the risk of NHL.

Materials and methods

Study population

Subjects for this population-based case–control study were recruited among female residents of Connecticut from 1995 to 2001. Incident cases were identified using the Yale Comprehensive Cancer Center's Rapid Case Ascertainment Shared Resource (RCA), a component of the Connecticut Tumor Registry (CTR). Eligible cases included female residents of Connecticut diagnosed with NHL (ICD-O, M-9590-9642, 9690-9701, 9740-9750), who were between the ages of 21 and 84 at the time of diagnosis, had no previous diagnosis of cancer (except non-melanoma skin cancer), and were alive at the time of the interview. The CTR is a population-based tumor registry that receives reports of cancer cases from both licensed hospitals and clinical laboratories, as mandated by Connecticut Public Health Code. RCA field staff survey all non-pediatric Connecticut hospitals in order to identify newly diagnosed cases. Demographic information on cases identified in the field is entered and verified by RCA data entry staff and screened against the CTR database. The registry also incorporates a system of reciprocal reporting with neighboring states and Florida to ensure complete case ascertainment. From 1995 to 2001, a total of 1122 potential NHL cases were identified. Of these 1122 potential cases, 290 were ineligible for this study (167 died before they could be interviewed, and 123 were excluded for other reasons, such as previous diagnosis of cancer, unable to speak English, or physician refusal). Thus, a total of 832 incident, eligible cases of NHL were contacted for participation in this study, and 601 (72%) of these cases completed in-person interviews. The median time between diagnosis and interview for cases was 2.5 months.

Histologic confirmation of cases was performed by two study pathologists (Drs Flynn, Tallini). Tissue samples were obtained from the pathology department where the case was diagnosed. Each sample was independently reviewed by both study pathologists and classified according to both the Working Formulation and World Health Organization (WHO) classification schemes [1]. Tissue samples receiving conflicting classifications were re-evaluated until a consensus was reached.

A population-based control group of women, aged 21–84, was assembled using two methods. Women less than 65 years of age were recruited using random digit dialing. Including the initial telephone screening, 69% of the women contacted using random digit dialing completed in-person interviews. Women 65 years of age and older were selected randomly from the files of the Centers for Medicare and Medicaid Services. Of the women contacted from these files, 47% completed in-person interviews. The number of controls that was randomly selected within each age stratum was adjusted every few months in order to frequency match cases by age within 5-year groups. A total of 718 controls completed in-person interviews.

Data collection

This study was conducted based on a protocol approved by the Human Investigations Committee at Yale University and the Connecticut Department of Health, and an Institutional Review Board of the National Cancer Institute. Cases were contacted only after approval by each subject's physician. Controls were contacted after selection through random sampling. All subjects were approached first by letter and then by telephone. Trained interviewers administered a standardized, structured questionnaire to subjects who agreed to participate, either at the subject's home or at a convenient location. During the interview, respondents were asked about their history of alcohol consumption and other known or suspected risk factors for NHL. Women were first asked whether they had ever consumed at least 12 drinks per year of each type of alcohol. If they had, they were further asked to provide information on the age they first drank, the duration and intensity of consumption, and whether they stopped drinking. One drink was considered to be one 12-ounce can or bottle of beer, one 4-ounce glass of wine, or one shot of liquor. Additional

information on age, height, usual weight, education, race, history of cigarette smoking, menopausal status, family history of cancer, diet, and other factors was obtained during the interview. Continuous demographic variables and potential confounding factors were categorized *a priori* based on previous cutpoints used in the literature or the distribution among study subjects.

Statistical analysis

Statistical analyses for this study were performed using the SAS system, version 8.02 (SAS Institute Inc., Cary, NC). Unconditional logistic regression models were developed using data on history of alcohol consumption in order to predict the risk of NHL and NHL subtypes [16].

Since women were questioned regarding consumption of each type of alcohol, separate analyses of the impact of beer, wine, and liquor on risk of NHL were performed. For each type of alcohol, the age first drank, intensity (g of ethanol per month), and duration (years) of consumption, and the total lifetime consumption (kg of ethanol) were considered. For example, women who reported consumption of less than 12 drinks of wine per year over their lifetime were classified as never drinkers of wine, and used as the reference group for analyses of the impact of wine consumption. The average monthly consumption of wine was calculated by multiplying the average number of days per month a subject reported consumption of wine by the average number of 4-ounce glasses of wine consumed on those days. This value was then multiplied by the ethanol content of wine (10.8 g of ethanol per 4-ounce glass) to determine the intensity of wine consumption. Finally, lifetime consumption of wine (kg) was calculated by multiplying the intensity and duration of wine consumption. Similar calculations were performed for analyses of beer and liquor consumption (beer: 13.2 g of ethanol per can or bottle; liquor: 15.1 g of ethanol per shot) [12]. All analyses by type of alcohol included dichotomous variables (ever/never) for consumption of the other two types of alcohol considered. Continuous predictor variables, including age at initiation, intensity, and duration of consumption, and total lifetime consumption were categorized *a priori* into tertiles based on the distribution among study subjects.

Self-reported data of consumption of beer, wine, and liquor were combined to estimate the overall impact of alcohol consumption. Women who reported consumption of less than 12 drinks per year of any type of alcohol at anytime during their lifetime were classified as never drinkers, and used as the reference group for analyses of all types of alcohol combined. The age at initiation of drinking was defined as the youngest reported age a subject began drinking beer, wine, or liquor. The duration of drinking was defined as the period of time during which subjects consumed any type of alcohol. The intensity of alcohol consumption (g of ethanol per month) and the lifetime estimate of ethanol consumption (kg of ethanol) were calculated by summing the contribution of each type of alcohol. As with each alcohol subtype, continuous predictor variables were categorized *a priori* into tertiles based on the distribution among study subjects.

Age, race, family history of cancer, education, body-mass index (BMI), pack-years of cigarette smoking, diet, and menopausal status were considered as potential confounding factors for this analysis. The associations of potential confounding factors with alcohol predictor variables and NHL subtypes were assessed using Pearson's χ^2 statistic. Decisions on which covariates to include in the final model were based on a greater than 10% change in the risk estimates for at least some NHL subtypes. Final estimates of the risk of NHL and NHL subtypes using the alcohol predictor variables were adjusted for age ($<$50, 50–70, $>$70 years) and education (high school or less, some college, college graduate or more). The inclusion of race (white, other), family history of cancer (any cancer, none), BMI ($<$25, 25–29.99, $\geq$30 kg/m^2), pack-years of cigarette smoking (non-smoker; 1–7, 8–14, 15–33, $\geq$34 pack-years), menopausal status (pre-menopausal, post-menopausal), daily fruit consumption ($<$1, 1–2, $>$2 servings), daily vegetable consumption ($<$1.5, 1.5–2.5, $>$2.5 servings), daily fat consumption ($\leq$54, 55–80, $>$80 g), daily protein consumption ($\leq$50, 51–66, $>$66 g), and daily animal protein consumption ($\leq$34, 35–50, $>$50 g) either individually or in groups did not result in a material change in the risk estimates, so these variables were excluded from the final model. Adjusted odds ratios (OR) and 95% confidence intervals (95% CI) were calculated using multivariate unconditional logistic regression models. Tests for linear trend were conducted by including alcohol variables as continuous variables in the multivariate unconditional logistic regression models.

Results

Selected demographic characteristics of cases and controls were compared (Table 1). Because of frequency matching, cases and controls were similar with respect to age, and the distribution of race and reported family history of cancer were also similar among cases and controls. However, cases tended to be less highly educated and have a higher BMI than controls.

Table 1. Selected characteristics of NHL cases and controls among women from Connecticut

	Percent of cases (n = 601)	Percent of controls (n = 718)
Age (years) ($\chi^2 = 0.74$, $p = 0.69$)		
<50	19.8	21.6
50–70	46.1	44.3
>70	34.1	34.1
Race ($\chi^2 = 0.60$, $p = 0.44$)		
White	95.3	94.3
Other	4.7	5.7
Family history ($\chi^2 = 2.20$, $p = 0.14$)		
None	21.5	24.9
Any cancer	78.5	75.1
Education ($\chi^2 = 12.26$, $p < 0.01$)		
High school or less	43.4	37.0
Some college	32.9	30.8
College graduate or more	23.6	32.2
BMI (kg/m^2) ($\chi^2 = 6.17$, $p = 0.05$)		
<25	49.8	56.5
25–29.99	31.6	27.9
≥30	18.6	15.6

Table 2 presents the risk of NHL by history of alcohol consumption. When compared to non-drinkers, women who reported consumption of at least 12 drinks per year over their lifetimes experienced a 20% decrease in risk of NHL (OR: 0.82, 95% CI: 0.65–1.04). In addition, increased duration of alcohol consumption was inversely associated with risk of NHL. Consumption of any type of alcohol for 25–40 years and greater than 40 years was associated with 10% (OR: 0.89; 95% CI: 0.65–1.22) and 40% decreased risk (OR: 0.62; 95% CI: 0.46–0.85), respectively, of NHL (p for linear trend $= 0.01$).

The slightly decreased risk of NHL associated with age at initiation, intensity of alcohol consumption, and lifetime alcohol consumption could be due to the relationships between these variables and the duration of alcohol consumption (Pearson's χ^2, $p < 0.01$). Stratification by duration of alcohol consumption revealed that the suggested inverse relationship between these variables and risk of disease only appeared among those subjects with longer durations of alcohol consumption (Table 3).

Controlling for other types of alcohol, analyses for each type of alcohol suggested that the inverse association between NHL and alcohol may be due to the consumption of wine (Table 4). Consumption of wine appeared to be inversely associated with risk of NHL (OR: 0.75; 95% CI: 0.59–0.96), while no association between NHL and consumption of beer (OR: 0.94; 95% CI: 0.71–1.23) or liquor (OR: 1.04; 95% CI: 0.81–1.33) was evident. However, if we had not controlled for the consumption of other types of alcohol in our analyses by

Table 2. Risk of NHL associated with alcohol consumption among women from Connecticut

	Cases #	Controls #	OR[a]	95% CI	p For linear trend[b]
Alcohol					
Never	230	233	1.00		
Ever	371	485	0.82	(0.65, 1.04)	
Age at initiation (years)					
≤19	124	168	0.81	(0.60, 1.11)	
20–24	123	152	0.87	(0.64, 1.18)	
≥25	124	165	0.79	(0.58, 1.06)	0.15
Intensity (g/month)					
<70	124	159	0.82	(0.61, 1.10)	
70–300	126	166	0.83	(0.62, 1.13)	
>300	121	160	0.82	(0.60, 1.10)	0.79
Duration (years)					
1–24	138	151	1.05	(0.76, 1.43)	
25–40	122	154	0.89	(0.65, 1.22)	
>40	111	180	0.62	(0.46, 0.85)	0.01
Lifetime consumption (kg)					
<20	132	164	0.85	(0.63, 1.15)	
20–70	115	152	0.82	(0.60, 1.12)	
>70	124	169	0.79	(0.59, 1.07)	0.68

[a] Adjusted for age and education.
[b] Test for linear trend conducted using continuous variable.

Table 3. Risk of NHL associated with alcohol consumption, stratified by duration of alcohol consumption, among women from Connecticut

	Duration 1–24 years		Duration 25–40 years		Duration >40 years	
	OR[a]	95% CI	OR[a]	95% CI	OR[a]	95% CI
Age at initiation (years)						
≤19	1.07	(0.65, 1.77)	0.97	(0.60, 1.58)	0.65	(0.41, 1.03)
20–24	1.26	(0.69, 2.30)	0.91	(0.58, 1.42)	0.77	(0.50, 1.18)
≥25	0.98	(0.66, 1.46)	0.82	(0.51, 1.33)	0.51	(0.30, 0.86)
p For linear trend[b]	0.89		0.43		<0.01	
Intensity (g/month)						
<70	0.97	(0.60, 1.55)	1.06	(0.65, 1.72)	0.64	(0.42, 0.99)
70–300	1.12	(0.71, 1.76)	0.89	(0.57, 1.39)	0.60	(0.37, 0.99)
>300	1.06	(0.67, 1.68)	0.77	(0.48, 1.24)	0.73	(0.45, 1.17)
p For linear trend[b]	0.90		0.68		0.73	
Lifetime consumption (kg)						
<20	0.99	(0.67, 1.47)	1.00	(0.62, 1.61)	0.61	(0.37, 1.03)
20–70	1.03	(0.63, 1.68)	0.87	(0.54, 1.39)	0.70	(0.43, 1.13)
>70	1.24	(0.71, 2.16)	0.84	(0.53, 1.31)	0.65	(0.43, 0.99)
p For linear trend[b]	0.34		0.36		0.51	

[a] Non-drinkers were used as the reference group for all analyses. Odds ratios were adjusted for age and education.
[b] Test for linear trend conducted using continuous variable.

Table 4. Risk of NHL associated with consumption of beer, wine, and liquor among women from Connecticut

	Beer		Wine		Liquor	
	OR[a,b]	95% CI	OR[a,c]	95% CI	OR[a,d]	95% CI
Alcohol						
Never	1.00		1.00		1.00	
Ever	0.94	(0.71, 1.23)	0.75	(0.59, 0.96)	1.04	(0.81, 1.33)
Age at initiation (years)						
≤19	0.96	(0.67, 1.37)	0.82	(0.55, 1.24)	0.99	(0.67, 1.46)
20–24	0.94	(0.61, 1.44)	0.78	(0.54, 1.11)	1.23	(0.88, 1.72)
≥25	0.89	(0.56, 1.43)	0.73	(0.55, 0.96)	0.92	(0.66, 1.28)
p For linear trend[e]	0.79		0.08		0.82	
Intensity (g/month)						
<70	0.96	(0.69, 1.34)	0.72	(0.55, 0.96)	1.07	(0.79, 1.44)
70–300	0.96	(0.61, 1.51)	0.73	(0.51, 1.05)	0.95	(0.64, 1.41)
>300	0.86	(0.52, 1.42)	0.90	(0.60, 1.37)	1.08	(0.72, 1.62)
p For linear trend[e]	0.47		0.63		0.76	
Duration (years)						
1–24	1.03	(0.72, 1.46)	0.90	(0.66, 1.22)	1.38	(1.00, 1.93)
25–40	1.03	(0.67, 1.58)	0.70	(0.49, 0.99)	1.01	(0.70, 1.45)
>40	0.68	(0.41, 1.13)	0.63	(0.44, 0.91)	0.69	(0.47, 1.02)
p For linear trend[e]	0.29		0.02		0.23	
Lifetime consumption (kg)						
<20	1.26	(0.87, 1.82)	0.66	(0.48, 0.91)	1.10	(0.78, 1.55)
20–70	0.71	(0.45, 1.12)	0.88	(0.63, 1.25)	1.06	(0.74, 1.54)
>70	0.80	(0.53, 1.22)	0.76	(0.55, 1.07)	0.98	(0.70, 1.37)
p For linear trend[e]	0.79		0.56		0.87	

[a] Adjusted for age and education.
[b] Also adjusted for consumption of wine (ever/never) and liquor (ever/never).
[c] Also adjusted for consumption of beer (ever/never) and liquor (ever/never).
[d] Also adjusted for consumption of beer (ever/never) and wine (ever/never).
[e] Test for linear trend conducted using continuous variable.

alcohol subtype, our results would have suggested a protective effect for all three types of alcohol, with the strongest association among wine drinkers (data not shown). Women reporting consumption of wine for 25–40 years and greater than 40 years was associated with 30% (OR: 0.70; 95% CI: 0.49–0.99) and 40% decreased risk (OR: 0.63; 95% CI: 0.44–0.91), respectively, of NHL (p for linear trend = 0.02).

The inverse association between wine consumption, but not beer or liquor consumption, and risk of NHL was consistent across all major NHL subtypes (data not shown). In addition, we found no statistically significant effect modification between alcohol consumption and cigarette smoking, family history of NHL, or family history of any cancer under the multiplicative model (data not shown).

Discussion

In this population-based case–control study of women in Connecticut, we found that consumption of alcohol appeared to be inversely associated with risk of NHL. Specifically, increased duration of wine consumption appeared to reduce risk of NHL, while consumption of beer and liquor did not seem to impact risk of NHL.

The observed inverse association between wine consumption and risk of NHL is consistent with several other recent epidemiologic studies [9, 11, 12, 14]. After controlling for consumption of beer and liquor, a small hospital-based case–control study in Uruguay suggested a weak inverse relationship between wine consumption and NHL among men [11]. In that study, male wine drinkers who consumed 1–60 and greater than 60 ml of ethanol per day experienced 20 and 30% reductions in risk, respectively; however, these results were not statistically significant, possibly due to the small sample size. A large population-based case–control study in the United States reported a 20 and 60% decrease in risk of NHL among men consuming 1–6 drinks/week and ≥1 drink/day of wine, respectively, after controlling for beer and liquor consumption [14].

A population-based case–control study in Los Angeles County reported a non-significant inverse association between alcohol consumption and risk of NHL among men and women [9]. The study suggested an inverse relationship for all three types of alcohol with NHL risk. However, the study failed to control for consumption of other types of alcohol when evaluating each alcohol type separately. The Iowa Women's Health Study, a prospective cohort study, reported a 22 and 40% significant decrease in risk with consumption of less than 3.4 g and greater than 3.4 g of alcohol consumed per day, respectively [12]. The study also found an inverse association for all three types of alcohol, however, as in the study in Los Angeles County, the authors did not control for consumption of other types of alcohol when specific types of alcohol were considered.

Our finding of a linear decrease in risk of NHL with increased duration of wine consumption has not been described before. Previous studies suggesting an inverse association between wine consumption and risk of NHL have not reported the impact of duration of consumption, but instead have considered only overall wine consumption and intensity [9, 11, 12, 14]. In addition, one study reported a decrease in risk with initiation of wine consumption earlier than 16 years of age [14]. In contrast, our study results suggest that duration of wine consumption is a more important predictor of NHL risk than intensity or age at initiation, since the associations between these variables and risk of NHL are only evident among those women with the longest duration of alcohol consumption (Table 3).

A potential inverse association between wine consumption and risk of NHL is supported by recent experimental studies of the immunomodulatory effects of wine [17, 18] and resveratrol [19–22], although the definite mechanism is currently unknown. Consumption of high levels of alcohol is known to suppress immune function [17, 18]. Experimental studies have shown that mice consuming ethanol exhibited significantly lower lymphocyte response and reduced activity of Phase I and Phase II enzymes involved in the detoxification of alcohol than mice consuming water [17]. However, mice consuming the same amount of ethanol in the form of red wine exhibited normal lymphocyte response and higher than normal cytochrome P450 activity [17]. The authors of that study hypothesized that phytochemicals present in red wine may be responsible for the protective immunomodulatory effects observed [17]. Experimental studies of resveratrol, a phytoalexin found in grape skins that is present in wine but not beer or liquor, has been shown to be a potent inhibitor of the initiation, promotion, and progression phases of tumorigenesis in both human leukemia and lymphoma cells [19–22]. If resveratrol does indeed play a role in reducing the risk of NHL, the observed reduction in risk of NHL with increased duration of wine consumption may be a result of the cumulative inhibition of resveratrol in multiple phases of tumorigenesis. Since resveratrol is found in grape skins, its concentration is higher in red wine than in white wine [19]. Although information on the type of wine consumed was not collected for this study, future studies of the potential protective role of wine in the development of NHL should distinguish between the

consumption of red and white wine, and incorporate measures of other beverages derived from grapes.

As mentioned previously, two epidemiologic studies have reported a positive association between overall alcohol consumption and risk of NHL [8, 15]. One of the studies involved a small cohort of men, and the relationship was observed only among those drinking beer and smoking cigarettes [8]. The other study, a population-based case–control study, reported an increased risk only among those with a family history of NHL [15]. Our data do not support a significant effect modification between alcohol consumption and cigarette smoking, family history of NHL, or family history of any cancer under the multiplicative model. Several hospital-based case–control studies found no association between alcohol drinking and risk of NHL [4–6, 10, 11, 13]. But, a lack of association in these studies could reflect the use of hospital-based controls, whose pattern of alcohol consumption may be related to the reason for hospitalization.

Although this study was population-based, the relatively low participation rate among potential population controls 65 years of age and older recruited from the files of the Centers of Medicare and Medicaid Services, are of possible concern. However, it is unlikely that selection bias can completely explain the observed results in our study for two reasons. First, the results were similar for women less than 65 years of age and women 65 years of age or older (data not shown). Second, the association between alcohol and NHL was specific to wine drinkers, and found only among those with the longest duration of consumption. Recall bias is possible, given the use of self-reported data on alcohol consumption. However, it is unlikely that this bias would be differential since the possible association between wine consumption and risk of NHL is not well known. Therefore, any recall error is likely to be random, which would result in an underestimation of the true association between wine consumption and risk of NHL. It is also possible that residual confounding could partially account for the observed inverse association between wine consumption and risk of NHL, given the relative paucity of knowledge surrounding the etiology of NHL. However, the inclusion of a number of suspected risk factors in the logistic regression models did not alter the observed risk estimates. The limited sample size resulting from stratification by both type of alcohol and subtype of NHL prevented a thorough analysis of the relationship between alcohol consumption and NHL subtypes. However, the inverse relationship between wine consumption and NHL appeared to be consistent across all major subtypes of NHL, regardless of the use of the Working Formulation or WHO classification scheme. Finally, it is unlikely that chance can account for the observed inverse relationship, given the consistency of the estimates for wine, but not beer or liquor.

A number of strengths of this study also should be considered in the interpretation of the results. In this large, population-based case–control study, data were gathered by trained interviewers using in-person, standardized, and structured interviews to minimize information bias resulting from exposure misclassification. Histologically confirmed, incident cases were categorized according to histologic type, immunologic type, and tumor grade in order to minimize information bias resulting from disease misclassification. In addition, subjects were asked standard, detailed questions regarding their lifetime history of alcohol consumption. Specifically, each subject was questioned separately for beer, wine, and liquor regarding the age at initiation, the number of years of consumption, the average number of days per month she drank and the average number of drinks consumed on those days, and whether she had stopped drinking. This detailed questioning for each type of alcohol allowed us to determine the potential impact of beer, wine, and liquor separately, which few other epidemiologic studies have done.

In summary, this study reports an inverse association between risk of NHL and increased duration of wine consumption. Our results are consistent with several recent epidemiologic studies, suggesting an inverse association between wine consumption and NHL risk. Other studies may have been inconsistent because they did not control for consumption of other types of alcohol when evaluating risk by type of alcohol. The results of this study highlight the importance of considering the type of alcohol consumed and the duration of consumption. Further research is needed to confirm these findings and explore the mechanism by which wine may affect NHL.

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